

Pneumoconioses

Vinaya S. Karkhanis and J.M. Joshi

Department of Pulmonary Medicine, T. N. Medical College and B.Y.L. Nair Hospital, Mumbai, India

ABSTRACT

Occupational lung diseases are caused or made worse by exposure to harmful substances in the work-place. "Pneumoconiosis" is the term used for the diseases associated with inhalation of mineral dusts. While many of these broad-spectrum substances may be encountered in the general environment, many occur in the work-place for greater amounts as a result of industrial processes; therefore, a range of lung reactions may occur as a result of work-place exposure. Physicians in metropolitan cities are likely to encounter pneumoconiosis for two reasons: (i) patients coming to seek medical help from geographic areas where pneumoconiosis is common, and (ii) pneumoconiosis caused by unregulated small-scale industries that are housed in poorly ventilated sheds within the city. A sound knowledge about the various pneumoconioses and a high index of suspicion are necessary in order to make a diagnosis. Identifying the disease is important not only for treatment of the individual case but also to recognise and prevent similar disease in co-workers.

[Indian J Chest Dis Allied Sci 2013;55:25-34]

Key words: Pneumoconiosis, Occupation, Fibrosis.

INTRODUCTION

History of mining goes far back to pre-historic times as far back as ancient Greece and Rome, when men mined for salts, flint and ochre. Inhalation of dust being hazardous was recognised since then and the diseases caused by it were labelled as asthma, Bergsucht disease (in which there is difficult breathing and short windedness with severe hard cough and marked hoarseness), silicosis, and pneumokoniosis, today what is known as pneumoconiosis. Occupational lung diseases, such as pneumoconiosis are mainly due to exposure to inorganic dust that is retained in the lung parenchyma and inciting fibrosis. In contrast, lung diseases due to organic dust are not associated with such accumulation of particles within the lungs and is believed to have an immunologic pathogenesis. Zenker in 1866 coined the term 'pneumonokoniosis'. Proust's in 1874 modified the term as 'pneumoconiosis' that means 'dusty lung'. It is defined for legal purposes as fibrosis of the lung due to silica dust, asbestos or other dusts and includes the condition known as dust reticulation. For medical purposes pneumoconiosis was described by Parkes as the presence of inhaled dust in the lungs and the non-neoplastic reaction to it.¹ Dusts that cause fibrosis are termed fibrogenic. It is important to differentiate those dusts that provoke a fibrotic reaction in the lung, from those that are retained

without such a reaction. Silicosis, coal worker pneumoconiosis, asbestosis, berylliosis and talcosis are examples of fibrotic pneumoconiosis. Siderosis, stannosis and baritosis are non-fibrotic forms of pneumoconiosis that result from inhalation of iron oxide, tin oxide, and barium sulfate particles, respectively.²

PATHOGENESIS³

After inhalation of dust, the alveolar macrophages converge upon extra-cellular particles and engulf them. If the number of particles is large, the elimination mechanism fails and dust containing macrophages collect in the interstitium especially in perivascular and peribronchiolar regions. If these aggregates remain *in situ*, type 1 pneumocytes grow over them so that they become enclosed and are then entirely interstitial in position. According to the amount of dust and cell accumulation the alveolar walls either protrude into the alveolar spaces or obliterate them. At the same time, a delicate supporting framework of fine reticulin fibers develops between the cells and in the case of dusts with fibrogenic potential, proliferation of collagen fibers follows. Inert dust such as carbon, iron, tin and titanium remain within the macrophages in these lesions until these cells die at the end of their normal life span. The particles are released and reingested by other macrophages. Some dust-laden macrophages

[Received: November 13, 2012; accepted after revision: December 14, 2012]

Correspondence and reprint requests: Dr J.M. Joshi, Professor and Head, Department of Pulmonary Medicine, T.N. Medical College and B.Y.L. Nair Hospital, Mumbai-400 008, India; Phone: 91-022-23027642/43; Fax: 91-022-23003095; E-mail: drjoshijm@email.com and drjoshijm@gmail.com

continually migrate to lymphatics or to bronchioles where these are eliminated. Migration is increased by infection or oedema of the lungs.

DIAGNOSIS

The clinical diagnosis of pneumoconiosis is usually based on an occupational history, chest radiographic findings and compatible pulmonary function tests.⁴ All patients who present with respiratory symptoms should have a work and environmental history recorded. As many occupational exposures to various dusts have their effect years after exposure,⁵ a life-time exposure history should be obtained covering all previous and current jobs. It is also important to ask about smoking history. Cigarette smoking is associated with an increased risk of silicosis and coal workers' pneumoconiosis. Smoking related symptoms and diseases may confound the presentation in pneumoconiosis. Patients with significant amount of disease will have symptoms of shortness of breath, cough, chest tightness, and/or wheezing. Those with less significant disease may not have any respiratory symptoms. Haemoptysis and fever may be the initial presentation where pulmonary tuberculosis develops as a complication of silica exposure. Presentation may uncommonly be with non-respiratory symptoms, such as symptoms of scleroderma or rheumatoid arthritis. Physical signs may be absent early in these diseases. There are no specific signs. Clubbing of fingers and toes may occur as the severity of the condition increases. Pulmonary function testing should be performed in all symptomatic patients.

Although the standard chest radiograph remains the mainstay of diagnosis of pneumoconiosis,⁶ several recent studies have confirmed that computed tomography (CT) has an increasing role in the radiologic evaluation of occupational lung disease.⁷ High-resolution CT (HRCT) is more sensitive than a chest radiograph for detection of parenchymal abnormalities in pneumoconiosis. It is well recognised that HRCT is the method of choice for patients with known or suspected infiltrative lung disease. Some controversy exists concerning the optimal CT technique for the evaluation of pneumoconiosis. HRCT is performed with thin collimation, i.e. 1-2 mm slice thickness, and a high spatial frequency algorithm. Slices are usually obtained at 10mm intervals from the lung apices to the diaphragm.⁸ When pneumoconiosis such as asbestosis are being investigated, prone scans must be obtained in order to distinguish dependent opacities at the bases from early pneumoconiosis. In pneumoconiosis, that are characterised by nodular opacities such as coal worker's pneumoconiosis and silicosis, there is some evidence to suggest that

conventional 10mm thick sections should also be obtained in addition to HRCT. Some investigators have recommended a combination of conventional CT, and HRCT scans to allow recognition of subtle parenchymal changes, such as minimal emphysema or mild lung fibrosis. In such instances, HRCT scans should be limited to a few slices at pre-selected levels. Contrast medium is not routinely utilised. CT and HRCT are not recommended for routine screening of exposed workers. However, CT may have a useful role in pneumoconiosis, particularly in the evaluation of the symptomatic worker with a normal chest radiograph. It may also be helpful in detecting another disease, that may be present, such as emphysema, and in distinguishing pleural from parenchymal disease. In several studies HRCT shown to have higher sensitivity and specificity compared to conventional chest radiography, in particular for asbestos-related diseases.¹⁵

The International Labour Organization (ILO)⁹ has established a standardised system for classifying radiographic abnormalities in pneumoconiosis as small-rounded opacities, small irregular opacities, zonal distribution of opacities and pleural thickening.⁹ This is described in table 1. Fluoro-2-deoxy-D-glucose positron emission tomography (FDG PET) scan can identify progressive massive fibrosis (PMF) lesions as hyper metabolic lesions even without associated malignancy or tuberculosis (TB).¹⁰ An open lung biopsy is rarely needed for the diagnosis.

TYPES OF PNEUMOCONIOSIS

The occupational lung disorders according to the biological properties of the material inhaled have been grouped into four main types¹¹: (i) disorders caused by exposure to mineral dust; (ii) disorders caused by exposure to gases and fumes; (iii) disorders caused by exposure to organic dust; and (iv) pulmonary and pleural malignancy caused by asbestos exposure leading to pleural mesothelioma. Pneumoconiosis represents a spectrum of pathological reactions of lung tissue to permanent deposition of inhaled particulate mineral dust or fibrous matter of occupational or environment origin. The severity of the disease is related to the material inhaled and the intensity and duration of the exposure. The pneumoconiosis primarily affects those exposed at work, but environmental exposure can affect others as well. It is characterised by non-neoplastic granulomatous and fibrotic changes in the lungs after the inhalation of inorganic substances, such as crystalline silica, asbestos, or coal dust.¹² Inorganic dusts are fibrogenic and non-fibrogenic. The pathological features of pneumoconiosis include lung nodules and fibrosis.¹³ Progression of pneumoconiosis is known to occur after the cessation

Table 1. Classification of radiographs of pneumoconioses

Features	Codes	Definitions	
Technical Quality			
	1	Good	
	2	Acceptable: with no technical defect likely to impair classification of the radiograph for pneumoconiosis	
	3	Acceptable: with some technical defect but still adequate for classification purpose	
	4	Unacceptable for classification purpose	
*If technical quality is not grade 1, a comment must be made about the technical defect			
Parenchymal Abnormalities			
Small opacities			
<i>Profusion:</i> The category of profusion is based on assessment of the concentration of opacities by comparison with the standard radiographs			
	0/-,0/0,0/1	Category 0: Small opacities absent or less profuse than category 1	
	1/0,1/1,1/2	Category 1	
	2/1,2/2,2/3	Category 2	All represent increasing profusion of small opacities as defined by the standard radiograph
	3/2,3/3,3/+	Category 3	
<i>Zones:</i> The right and left thorax are divided into three zones: upper (U), Middle (M), and lower (L) as RU, RM, RL and LU, LM, LL			
<i>Shape and size:</i> The letters p, q, and r denote the presence of small rounded opacities, with three sizes defined by the appearances as: P= diameter up to about 1.5mm q= diameter exceeding about 1.5mm to 3mm r= diameter exceeding about 3mm to 10mm			
Rounded irregular Mixed	p/p, q/q, r/r s/s, t/t, u/u	For mixed the predominant shape and size is recorded first and significant number of another shape and size is recorded after the oblique stroke	
Large opacities: An opacity having longest dimension exceeding 10mm			
	0,A,B,C	Category O: No large opacity	
		Category A: One large opacity with longest diameter up to about 50mm or several large opacities with the sum of their longest diameter not exceeding about 50mm	
		Category B: One large opacity having the longest dimension exceeding 50mm but not exceeding the equivalent area of the right upper zone, or several large opacities with the sum of their longest dimensions exceeding 50mm but not exceeding the equivalent area of the right upper zones	
		Category C: One large opacity which exceeds the equivalent area of the right upper zone, or several large opacities which when combined exceed the equivalent area of the right upper zone	
Pleural Thickening			
<i>Pleural Plaques or diffuse (chest wall/ diaphragm)</i>	Plaques involving diaphragmatic pleura are recorded separately as present or absent		
Site width	R, L	Right or left	
	R (a, b, c) L(a, b, c)	a: maximum width up to 5 mm b: maximum width over about 5mm to 10mm c: maximum width over 10mm	
Extent (Defined in terms of maximum length of pleural involvement)	R(1, 2, 3) L(1, 2, 3)	1: Total length equivalent to up to 1/4 of the projection of the lateral chest wall 2: Total length exceeding ¼ but not one half of the projection of the lateral chest wall 3: Total length exceeding one half of the projection of the lateral chest wall	
<i>Costophrenic angle obliteration:</i> It is recorded as absent or present and the site as right or left			
<i>Pleural calcification:</i> Site and extent are recorded for 2 lungs separately as right or left			
Adapted from: Guidelines for the use of the ILO international Classification of radiographs of pneumoconioses. Occupational safety and health series no. 22 (revised edition 2011).			

of dust exposure.¹⁴ In recent years the use of HRCT has proved very reliable for the detection of occupationally induced pneumoconiosis. Depending on the tissue reaction caused by the dust, pneumoconiosis can be further classified as shown in table 2.

Table 2. Classification of pneumoconiosis

Type	Dust / Type	Radiograph
Pneumoconiosis mimic (inert dust)	Siderosis (iron dust) Stannosis (tin dust) Baritosis (barium sulfate)	Chest radiograph shows opacities due to retention of the dust but there is no fibrosis or functional abnormality
Uncomplicated pneumoconiosis (without PMF)	Alluminosis (aluminium) Talcosis (talc) Berylliosis (beryllium)	Dusts cause fibrosis but no PMF
Complicated pneumoconiosis (with PMF)	Coal worker's pneumoconiosis (coal) Silicosis (silica) Asbestosis (asbestos)	Dusts causing fibrosis with PMF.

PMF=Progressive massive fibrosis

Pneumoconiosis Mimics/Inert Dust Pneumoconiosis

Inert dusts are inorganic in origin and neither cause proliferation of reticulin fibres nor give rise to collagenous fibrosis when retained in the lungs. The various entities are described below.

Siderosis

Siderosis (buffer's lung or silver polisher's lung) is a non-fibrogenic or a "benign" form of pneumoconiosis due to inhalation of iron particles. It is caused by the accumulation of iron oxide in macrophages within the lung.² Dust or fumes of metallic iron oxide encountered in various processes like iron and steel rolling mills, steel grinding, electric arc welding, silver polishing, mining and crushing iron ores are responsible for producing this kind of pneumoconiosis. It is observed most commonly in workers exposed to metal fumes during welding, and thus, is also known as welder siderosis or arc welder pneumoconiosis. Doig and McLaughlin¹⁶ first described 'welders' siderosis in 1936 when they carried out a prospective study examining the clinical and chest radiological characteristics of 16 electric arc welders. Though non-fibrogenic fibrosis is seen in some occasionally and is likely due to associated silica exposure causing silicosiderosis.¹⁷ Pathologically, the cut-surface of the lung reveals grey to brown coloured macules from 1mm to 4mm in diameters and these do not stand up from the surface. These are evenly distributed and can regress. Microscopically, the lesion consists of a perivascular and peribronchiolar aggregation of dark pigmented iron oxide particles present in macrophages and alveolar spaces and walls. Slight reticulin proliferation may be present

but there is no collagenous fibrosis. Clinically these may be reddish-coloured sputum. No other symptoms or physical signs are present. Usually, there is no impairment of lung function. However, many surveys among welders have shown the association of welding with obstructive airway

diseases as severe as that related to smoking.¹⁸ The chest radiograph shows a variable, usually, large number of small nodular opacities varying from 0.5mm to 2mm in diameter with fine linear opacities. Kerley's B lines may be present in some patients caused by the accumulation of iron in interlobular septa. The hilar nodes may appear unusually radio-opaque due to the concentrated iron content; but these are not enlarged. The HRCT shows widespread, ill-defined, small centrilobular nodules and, less commonly, patchy areas of ground-glass attenuation without zonal predominance.¹⁹ Emphysema is often seen. After cessation of exposure, the iron dust is slowly eliminated from the lungs over a period of years. This results in the partial or complete disappearance of radiographic opacities.²⁰

Stannosis

Stannosis is a condition in which tin-oxide is deposited in the lung tissue after inhalation. It was first recognised in Germany during the Second World War. Occupational stannosis in non-mining industry may occur with earth tinning where molten tin is poured into heated iron hollow-ware or articles are dipped by hand into molten tin to coat them. Grinding, briquet-making, smelting and casting of tin, as well as the handling of tin-oxide in industry may cause stannosis. Stannosis in tin mines is caused by the process of bagging where highly concentrated (70%-80%) tin-oxide is packaged for transport into a tin smelter and is a very dusty occupation.²¹ Cut-surface of the lungs reveals numerous tiny (1mm-3mm), grey-black dust macules, soft to touch and not raised above the cut-surface of the lung. Macrophages containing tin-oxide dust particles are present in alveolar walls and spaces,

perivascular lymphatics and interlobular hilar nodes. There are no symptoms and abnormal physical signs due to the inhalation and retention of tin-oxide dust. Lung function is unaffected.²² Radiologically, it presents with numerous small, very dense opacities scattered evenly throughout the lung fields and may even be somewhat larger at 2mm to 4mm diameter and more fluffy or irregular in outline than those of siderosis. Kerley's lines are often clearly defined and dense linear opacities are seen in the upper lung zones.⁸

Baritosis

Baritosis, a benign type of pneumoconiosis, is caused by long-term exposure to barium dust. It was first described by Fiori in Italy. Inhaled particulate matter remains in the lungs for years without producing symptoms, abnormal physical signs or interference with lung function. Owing to the high radio-opacity of barium, the discrete shadows in the chest radiograph are extremely dense.²³ The discrete opacities in baritosis clear slowly over the years.²⁴

Uncomplicated Pneumoconiosis (Figure 1)

Pneumoconiosis that causes fibrosis in the lung parenchyma but without PMFs are termed as 'uncomplicated pneumoconiosis'.



Figure 1. High resolution computed tomography of chest showing simple pneumoconiosis.

Aluminosis

Lung diseases induced by aluminium dust are very rare. Pulmonary aluminosis is a disease first seen in Germany between 1938 and 1945 which then noticed in the United Kingdom between 1952 and 1959.²⁵ Inhalation of dusts containing metallic and oxidised aluminum is associated with the development of pulmonary fibrosis. It may be caused by concomitant exposure to free silica and not to the

aluminum oxide. Desquamative interstitial pneumonia,²⁶ a granulomatous lung reaction and pulmonary alveolar proteinosis may occur after exposure to aluminium fumes.²⁷ Histological examination of lung tissue samples shows severe subpleural and interstitial fibrosis with scar emphysema and spotted granulomatous pneumonitis with giant cells. Aluminium lung is characterised as diffuse interstitial fibrosis that is mainly located in the upper and middle lobes of the lung.²⁸ In advanced stages, it is characterised by subpleural bullous emphysema with an increased risk of spontaneous pneumothorax. The HRCT findings include small, centrilobular, nodular opacities and slightly thickened interlobular septae.²⁹ As a metal powder, the element also has unusual and unexplained properties that may modify the course of pulmonary fibrosis in silicosis.

Talcosis

Talc is a mineral widely used in the ceramic, paper, plastics, rubber, paint, and cosmetic industries. Talcosis, a granulomatous inflammation of the lungs caused by inhalation of talcum dust, is a rare form of silicate induced lung disease. Thorel³⁰ reported the first case of talc pneumoconiosis in 1896. Talc has been recognised as a cause of pneumoconiosis in miners, millers, rubber workers.³¹ Talc exposure may occur as a result of inhalation or by intravenous administration. Three forms of talc pneumoconiosis by inhalation have been described in the literature: talc asbestosis, talc silicosis, and talcosis. Patients often present with isolated, non-specific symptoms of progressive exertional dyspnoea or cough. Histologically, it causes non-necrotising granulomatous inflammation characterised by the formation of foreign body granulomas of varying degree within a fibrotic stroma. These granulomas are composed of free or intracellular birefringent deposits accompanied by multinucleated giant cells. These may also appear ill defined with only few surrounding histiocytes. The distribution of these lesions is variable and these may develop in intra- and peri-vascular areas as well as in the interstitium.³² Chest radiography shows micronodular patterns of diffuse or well-defined nodules that may fuse with progression to form larger opacities in the perihilar regions. The HRCT findings of small centrilobular nodules associated with heterogeneous conglomerate masses containing high-density amorphous areas, with or without panlobular emphysema in the lower lobes, are highly suggestive of pulmonary talcosis. Interstitial thickening and lower-lobe emphysema may also be present.³³

Berylliosis

Berylliosis, or chronic beryllium disease, is a chronic allergic-type lung response and chronic lung disease caused by exposure to beryllium and its compounds. Acute beryllium disease causes non-specific inflammatory reaction. Exposure to beryllium occurs in a variety of industries, including aerospace, ceramics, dentistry and dental supplies, nuclear weapons etc.³⁴ Beryllium exposure can lead to an acute chemical pneumonitis and a chronic granulomatous disease. Histologically, chronic beryllium disease may mimic sarcoidosis. The chest radiograph shows hilar lymphadenopathy and increased interstitial markings. On HRCT, the most common findings are parenchymal small nodules that are often clustered around the bronchi, interlobular septa, or in the subpleural region where the nodules may form pseudoplaques and interlobular septal thickening. Ground-glass opacities, honeycombing, conglomerate masses, bronchial wall thickening and hilar or mediastinal lymph nodes with amorphous or eggshell calcification may also be seen. Blood beryllium lymphocyte proliferation test (BeLPT) currently is the test of choice to identify beryllium workers who develop beryllium sensitisation or chronic beryllium disease.³⁵

Complicated Pneumoconiosis

The pneumoconiosis are associated with progressive massive fibrosis (Figures 2 and 3).



Figure 2. Chest radiograph (postero-anterior view) showing complicated pneumoconiosis with necrotic progressive massive fibrosis.

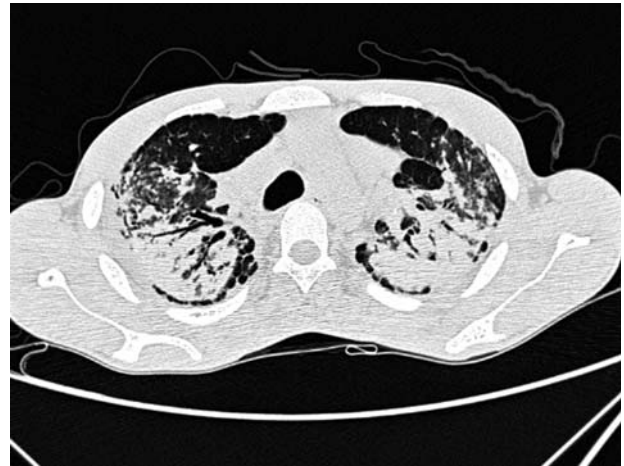


Figure 3. High resolution computed tomography of chest showing complicated pneumoconiosis with progressive massive fibrosis.

Silicosis

Silicosis is a fibrotic disease of the lungs caused by inhalation of dust containing free crystalline silica. In 1870, Visconti introduced the term silicosis, derived from the Latin word *silex*, or flint. Mining, tunneling, sand stone industry, stone quarrying and dressing, iron and steel foundries and flint crushing are the occupations most closely related to the hazards of silica exposure. Three different types of tissue reactions have been distinguished: (1) chronic, (2) accelerated and (3) acute. The 'chronic' is the most usual form of silicosis occurs after many years of exposure to relatively low levels of dust. It is rare for the chest radiograph to show any abnormality before 20 years of exposure. Silicosis is characterised by the presence of small discrete nodules exclusively distributed in the upper zones of the lung with a posterior predominance. These nodules tend to coalesce and form massive fibrotic lesions. PMF is seen as irregularly shaped masses, most frequently in apical and posterior segments of the upper and lower lobes, with peripheral parenchymal distortion. Various types of calcifications in PMF can be observed including punctuate, linear, or massive. Hilar and mediastinal lymphnodes tend to enlarge and calcify in an eggshell, punctate or massive form. Silicosis related lesions are assessed radiologically according to the ILO classification of pneumoconiosis. The HRCT at an early stage demonstrate irregular non-septal small linear opacities in addition to nodular opacities diffusely distributed throughout both the lungs. Serial CT scans demonstrate the progression of the disease from a diffuse linear pattern to a confluence of nodules with or without PMF. The HRCT is more sensitive than chest radiography in detecting lung parenchymal changes suggestive of silicosis and early confluence of small opacities in the lung.³⁶

The association between silicosis and pulmonary tuberculosis has been well established.³⁷ It has been suggested that the bacilli may be encapsulated in the silicotic nodules and cause an increased risk of future reactivation of the disease, or that the altered immunological profile in the lungs of the patients with silicosis might predispose a person to tubercular infection.³⁸ Cavitation may be seen in silicosis. Although cavitation in silicosis may occur without a mycobacterial infection, the latter should be considered in such a presentation. Silicosis is associated with an excessive decline in lung function. A tuberculin skin test using purified protein derivative (PPD) is indicated in all silicosis patients. If the test result is positive, treatment for latent or active infection is indicated.³⁹ In more advanced disease, airflow obstruction or a mixed pattern of obstruction and restriction is observed.

Coal Worker's Pneumoconiosis

Coal worker's pneumoconiosis (CWP) can be defined as the accumulation of coal dust in the lungs and the tissue's reaction to its presence.⁴⁰ The disease is divided into two categories: simple coal worker's pneumoconiosis (SCWP) and complicated coal worker's pneumoconiosis (CCWP), or PMF.⁴¹ Pneumoconiosis complicating rheumatoid arthritis has been described.^{42,43} Honma and Vallyathan⁴² classified rheumatoid pneumoconiosis into two types: classic (Caplan's syndrome), as described by Caplan⁴³ in 1953/1959, and by Gough in 1955 and silicotic, without rheumatoid nodules in the lungs, although with small silicotic nodules. These likely have an immunological basis. Radiographs remain the main diagnostic tool. The ILO's 12-point classification is used for standardising the diagnosis of coal worker's pneumoconiosis. The radiographic pattern of CWP, SCWP typically consists of small round nodular opacities and occasionally includes reticular or reticulonodular opacities. The risk of TB is increased in individuals with coal worker's pneumoconiosis, as it is in those affected by silicosis. Coal dust inhalation is also related to the development of chronic obstructive pulmonary disease, that contributes to increased mortality among these patients.

Asbestosis

The word asbestos is derived from Greek and means 'inextinguishable'. In the 1930s and 1940s, scientists recognised a causal link between asbestos exposure and asbestosis. The incidence of asbestosis varies with the cumulative dose of inhaled fibers; the greater the cumulative dose, the higher is the incidence of asbestosis. All types of asbestos fibers are fibrogenic to the lungs provided the individual has sufficient

exposure.⁴⁴ However, recent studies have shown that lower levels can also cause disease in some workers. Fibers with diameters less than 3µm are fibrogenic because these penetrate cell membranes. Smokers have an increased rate of progression of asbestosis; likely due to impaired mucociliary clearance of asbestos fibers⁴⁵. As the development of asbestosis is dose-dependent, symptoms appear only after a latent period of 20 years or longer. This latent period may be shorter after intense exposure. The latency between exposure onset and disease is wide, ranging from 15 years to more than 40 years. Dyspnoea upon exertion is the most common symptom and worsens as the disease progresses. Patients may have a dry (i.e., non-productive) cough. Finger clubbing is observed in 32%-42% of cases. Typical findings include diffuse reticulonodular infiltrates, that are observed predominantly at the lung bases. The HRCT allows a better definition of interstitial infiltrates and may be helpful in diagnosing asbestosis in the early stages. Typical HRCT findings in asbestosis include subpleural linear opacities seen parallel to the pleura; basilar lung fibrosis and peribronchiolar, intralobular, and interlobular septal fibrosis; honeycombing; and pleural plaques.⁴⁶ The American College of Pathologists' scheme for assessing the severity of asbestosis grades fibrosis in the following four categories:

Grade 1 - Fibrosis in the wall of a respiratory bronchiole without extension to distant alveoli

Grades 2 and 3 - These define more extensive disease

Grade 4 - Alveolar and septal fibrosis with spaces larger than alveoli, ranging up to 1cm (i.e., honeycombing)

Hard Metal Disease

Respiratory diseases in the hard metal industry have been recognised as a probable occupational hazard since 1940. Hard metal is an alloy of tungsten carbide, cobalt and occasionally other metals, such as titanium, tantalum, chromium, nickel.⁴⁷ Of these, cobalt whether free or in alloy form, is allergenic and cytotoxic and capable of provoking release of a fibrogenic agent from macrophages. Hard metal is generally produced by compacting powdered tungsten carbide and cobalt into a polycrystalline material, a process called sintering, hence the term 'sintered carbides'. This composite material has hardness almost like that of diamond; and is used to make machine parts that require high heat resistance, or to make tools used for drilling, cutting, machining, or grinding. The disease has been known by various names, such as hard metal pneumoconiosis, tungsten carbide pneumoconiosis, hard metal lung, giant cell interstitial pneumonitis, and cobalt lung.⁴⁸ Three

types of disorder are attributed to cobalt exposure: acute (in the form of asthma), sub acute (fibrosing alveolitis) and chronic in the form of diffuse interstitial fibrosis of the giant cell variety. One important aspect of hard-metal lung disease is that the disease may occur after a short duration of exposure, thus suggesting that individual susceptibility, rather than cumulative exposure, plays a major role.⁴⁹ A recent case series from the United States described the spectrum of respiratory diseases associated with exposure to hard metal, ranging from reversible airway obstruction to reversible hypersensitivity pneumonitis, alveolitis and pulmonary fibrosis.⁵⁰ The diagnostic criteria for hard-metal lung disease include the following: (a) a history of exposure to metal dust; (b) characteristic clinical features, including shortness of breath, cough, and dyspnea on exertion over a prolonged period; (c) radiologic findings of interstitial lung disease; (d) histologic findings of interstitial lung disease or a giant cell interstitial pneumonia pattern (presence of a large number of giant cells filling the airspace), with thickening of the interstitium and alveolar walls by mononuclear cells; and (e) a histopathologic finding of metallic content in lung tissue.⁵¹

Mixed-dust Fibrosis

Clinical diagnosis of mixed-dust fibrosis requires the exclusion of other well-defined pneumoconioses. Mixed-dust pneumoconiosis is defined as a pneumoconiosis caused by concomitant exposure to silica and less fibrogenic dusts, such as iron, silicates and carbon. The silica is usually at a lower concentration than what causes silicotic nodules to occur. Microscopically, a stellate shape characterises the mixed-dust fibrotic nodule and has a central hyalinised collagenous zone surrounded by linearly and radially arranged collagen fibers admixed with dust-containing macrophages. Generally, as the proportion of silica increases, the number of silicotic nodules increases in proportion to the mixed-dust nodules. The radiological findings of a mixed-dust pneumoconiosis include a mixture of small, rounded and irregular opacities. Honeycombing is also seen.⁵² Similar radiological changes were described earlier as 'Transkei silicosis' in females in Transkei region of South Africa by Palmer where maize is ground using silica containing rocks.⁵³ Subsequently the term 'hut lung' was coined by Grobbelaar and Bateman in patients exposed to components of organic maize and biomass fuel with non quartz containing dust for cooking in poorly ventilated room.⁵⁴ Sundaram *et al*⁵⁵ described cases of mixed-dust fibrosis that occurred in the setting of poorly ventilated flour mills where various kinds of grain, chiefly wheat, were ground using stones containing greater than 80% silica and

proposed the term 'Flour mill lung' (Figures 4 and 5) for this form of pneumoconiosis.



Figure 4. Chest radiograph (postero-anterior view) of a flour mill worker.



Figure 5. High resolution computed tomography of a flour mill worker.

Flock Worker's Lung

Flock worker's lung is an industrial lung disease. It is exclusive to employees of the rotary cut synthetic materials industry. Rotary-cut nylon, polyester, rayon and other synthetic fibers produce a powder of short

fibers that are then adhesive coated to fabrics and other objects to produce a velvety surface. Flock production is associated with an increased risk in workers of developing a chronic interstitial lung disease characterised by a lymphocytic bronchiolitis, bronchiolocentric nodular and diffuse lymphocytic interstitial infiltrates, and variable interstitial fibrosis.⁵⁶ The chest radiographs in flock worker's lung may reveal diffuse patchy infiltrates. HRCT may show scattered areas of consolidation, patchy ground-glass opacity and peripheral honeycombing.⁵⁷ HRCT is the most sensitive non-invasive tool for detecting flock worker's lung in the early stages.¹⁸

CONCLUSIONS

The diagnosis of pneumoconiosis can be made difficult by years of latency between exposure in the work-place and the occurrence of the disease. The severity of the disease is related to the material inhaled, and, the intensity and duration of the exposure. A sound knowledge about the various pneumoconioses and a high index of suspicion are necessary in order to make an appropriated diagnosis, especially in geographic areas where pneumoconiosis is uncommon. Identifying disease is important not only for the individual case but also to recognise and prevent disease in co-workers.

REFERENCES

- Fourth International Pneumoconiosis Conference. Report of the working party on the Definition of Pneumoconiosis. Geneva: International Labour Organization; 1971.
- Chong S, Lee KS, Chung MJ, Han J, Kwon OJ, Kim TS. Pneumoconiosis: comparison of imaging and pathologic findings. *Radiographics* 2006;26:59-77.
- Fujimura N. Pathology and pathophysiology of pneumoconiosis. *Curr Opin Pulm Med* 2000;6:140-4.
- Beckett WS. Occupational respiratory diseases. *N Engl J Med* 2000;342:406-12.
- Fraser P. Pleuropulmonary disease caused by inhalation of inorganic dust. In: Fraser P, editors *Diagnosis of Diseases of the Chest*; 3rd edition. Philadelphia: W.B. Saunders; 1990:pp2276-81.
- Pham QT. Chest radiography in the diagnosis of pneumoconiosis. *Int J Tuberc Lung Dis* 2001;5:478-82.
- Mcloud T. Occupational lung disease. *Radiol Clin North Am* 1991;29:931-41.
- Akira M. High-resolution CT in the evaluation of occupational and environmental disease. *Radiol Clin North Am* 2002;40:43-59.
- International Labour Organization (ILO). Guidelines for the use of ILO International Classification of Radiographs of Pneumoconiosis. Geneva: ILO; 1980.
- Chung SY, Lee JH, Kim TH, Kim SJ, Kim HJ, Ryu YH. 18F-FDG PET imaging of progressive massive fibrosis. *Ann Nucl Med* 2010;24:21-7.
- Crofton J, Douglas A. Occupational lung disease. In: *Respiratory Diseases*. Singapore: PG Publishing Pvt Ltd;1983:p.575.
- Kuschner WG, Stark P. Occupational lung disease. Part 2. Discovering the cause of diffuse parenchymal lung disease. *Postgrad Med J* 2003;113:81-8.
- Feng Y, Yang Z. Pneumoconiosis: high-resolution CT characteristics and patho-physiology. *Sheng Wu Yi Xue Gong Cheng Xue Za Zhi* 2010;27:219-21.
- Kimura K, Ohtsuka Y, Kaji H, Nakano I, Sakai I, Itabashi K, et al. Progression of pneumoconiosis in coal miners after cessation of dust exposure: a longitudinal study based on periodic chest x-ray examinations in Hokkaido. *Japanese Intern Med* 2010;49:1949-56.
- Aberle DR, Gamsu R, Ray CS, Feuerstein IM. Asbestos-related pleural and parenchymal fibrosis: detection with high-resolution CT. *Radiology* 1988;166:729-34.
- Doig AT, McLaughlin AIG. X-ray appearances of the lungs of electric arc welders. *Lancet* 1936;1:771-5.
- McCormick L, Goddard M, Mahadeva R. Pulmonary fibrosis secondary to siderosis causing symptomatic respiratory disease: a case report. *J Med Case Rep* 2008;2: 257.
- Billings CG, Howard P. Occupational siderosis and welders' lung: a review. *Monaldi Arch Chest Dis* 1993;48: 304-14.
- Flors L, Domingo ML, Leiva-Salinas C, Mazón M, Roselló-Sastre E, Vilar J. Uncommon occupational lung diseases: high-resolution CT findings. *AJR Am J Roentgenol* 2010;194:W20-6.
- Kim KI, Kim CW, Lee MK, Lee KS, Park CK, Choi SJ, et al. Imaging of occupational lung disease. *Radiographics* 2001;21:1371-91.
- Sluis-cremer GK, Thomas RG, Goldstein B, Solomon A. Stannosis: a report of 2 cases. *S Afr Med J* 1989;75:124-6.
- Yilmaz A, Göçmen Ocal S, Doruk S, Acu B. Is tin fume exposure benign or not: two case reports. *Tuberk Toraks* 2009;57:422-6.
- Doig AT. Baritosis: a benign pneumoconiosis. *Thorax* 1976;31:30-9.
- Wende E. *Pneumokoniose bei Baryt- und Lithoponarbeitern*. *Archiv fur Gewerbepathologie und Gewerhygiene* 1956; 15:171.
- Dinman BD. Aluminum in the lung: the pyropowder conundrum. *J Occup Med* 1987;29:869-76.
- Herbert A, Sterling G, Abraham J, Corrin B. Desquamative interstitial pneumonia in an aluminum welder. *Human Pathol* 1982;13:694-9.
- Voisin C, Fisekci F, Buclez B, Didier A, Couste B, Bastien F. Mineralogical analysis of the respiratory tract in aluminiumoxide-exposed workers. *Eur Respir J* 1996; 9:1874-9.
- Nasiadek M, Sapota A. Toxic effect of dust and fumes of aluminium and its compounds on workers' respiratory tract. *Med Pract* 2004;55:495-500.
- Kraus T, Schaller KH, Angerer J, Hilgers RD, Letzel S. Aluminosis—detection of an almost forgotten disease with HRCT. *J Occup Med Toxicol* 2006;17:1-4.
- Thorel C. Talc lung: a contribution to the pathological anatomy of pneumoconiosis. *Beitr Pathol Anat Allgem Pathol* 1896; 20:85-101.
- Vallyathan NV, Craighead JE. Pulmonary pathology in workers exposed to non asbestiform talc. *Human Pathol* 1981;12:28-35.
- Scheel AH, Krause D, Haars H, Schmitz I, Junker K. Talcum induced pneumoconiosis following inhalation of adulterated marijuana: a case report. *Diagn Pathol* 2012; 15:26.
- Marchiori E, Lourenço S, Gasparetto TD, Zanetti G, Mano CM, Nobre LF. Pulmonary talcosis: imaging findings. *Lung* 2010;188:165-71.

34. Lynch DA. Imaging of beryllium-related diseases. In: Genevois A, De Vust P, editors. *Imaging of Occupational and Environmental Disorders of the Chest*. Germany: Springer-Verlag; 2006:pp249-56.
35. Barna BP, Culver DA, Yen-Lieberman B, Dweik RA, Thomassen MJ. Clinical application of beryllium lymphocyte proliferation testing. *Clin Diagn Lab Immunol* 2003;10:990-4.
36. Remy-Jardin M, Remy J, Farre I, Marquette CH. Computed tomographic evaluation of silicosis and coal worker's pneumoconiosis. *Radiol Clin North Am* 1992;30:1155-76.
37. Snider DE. Relationship between tuberculosis and silicosis [editorial]. *Am Rev Respir Dis* 1978;118:455-60.
38. Iyer R, Holian A. Immunological aspects of silicosis. In: Castranova V, Wallyathan V, Wallace E, editors. *Silica and Silica-induced Lung Diseases*. Boca Raton: CRC Press; 1996:pp253-67.
39. Barboza CE, Winter DH, Seiscento M, Santos Ude P, Terra Filho M. Tuberculosis and silicosis: epidemiology, diagnosis and chemoprophylaxis. *J Bras Pneumol* 2008;34:959-66.
40. Morgan WK, Seaton A. *Occupational Lung Diseases*. Philadelphia: W.B. Saunders;1975:pp149-210.
41. Wade WA, Petsonk EL, Young B, Mogri I. Severe occupational pneumoconiosis among West Virginian coal miners: one hundred thirty-eight cases of progressive massive fibrosis compensated between 2000 and 2009. *Chest* 2011;139:1458-62.
42. Honma K, Vallyathan V. Rheumatoid pneumoconiosis: a comparative study of autopsy cases between Japan and North America. *Ann Occup Hyg* 2002;46:265-7.
43. Caplan A. Certain unusual radiological appearances in the chest of coal-miners suffering from rheumatoid arthritis. *Thorax* 1953;8:29-37.
44. American Thoracic Society. Medical section of the American Lung Association: the diagnosis of nonmalignant diseases related to asbestos. *Am Rev Respir Dis* 1986;134:363-8.
45. Churg A, Stevens B. Enhanced retention of asbestos fibers in the airways of human smokers. *Am J Respir Crit Care Med* 1995;151:1409-13.
46. Copley SJ, Wells AU, Sivakumaran P, Rubens MB, Lee YC, Desai SR, et al. Asbestosis and idiopathic pulmonary fibrosis: comparison of thin-section CT features. *Radiology* 2003;229:731-6.
47. Davison AG, Haslam PL, Corrin B, Coutts II, Dewar A, Riding WD, et al. Interstitial lung disease and asthma in hard metal workers: bronchoalveolar lavage, ultra structural and analytical findings and results of bronchial provocation tests. *Thorax* 1983;38:119-28.
48. Nemery B, Verbeke EK, Demedts M. Giant cell interstitial pneumonia in hard metal lung disease, cobalt lung. *Semin Respir Crit Care Med* 2001;22:435-47.
49. Nemery B, Bast A, Behr J, Borm PJ, Bourke SJ, Camus PH, et al. Interstitial lung disease induced by exogenous agents: factors governing susceptibility. *Eur Respir J* 2001;18:30s-42s.
50. Cugell DW, Morgan WKC, Perkins DG, Rubin A. The respiratory effects of cobalt. *Arch Intern Med* 1990;150:177-83.
51. Coates EO Jr, Watson JH. Diffuse interstitial lung disease in tungsten carbide workers. *Ann Intern Med* 1971;75:709-16.
52. Raymond PW. Non-neoplastic disorders due to metallic, chemical and physical agents. In: Raymonds PW, editor. *Occupational Lung Diseases*; 2nd edition. London: Butterworths;1982:pp464-66.
53. Palmer PES. Transkei silicosis. *South Afr Med J* 1967;41:1182-8.
54. Grobbelaar JP, Bateman ED. Hut lung: a domestically acquired pneumoconiosis of mixed etiology in rural woman. *Thorax* 1991;46:334-40.
55. Sundaram P, Kamat R, Joshi JM. Flour mill lung: a pneumoconiosis of mixed aetiology. *Indian J Chest Dis Allied Sci* 2002;44:199-201.
56. Gothi D, Joshi JM. Rayon Lung in a non-flocking industry worker. *Indian J Chest Dis Allied Sci* 2009;51:177-9.
57. Kern DG, Kuhn C 3rd, Ely EW, Pransky GS, Mello CJ, Fraire AE, et al. Flock worker's lung: broadening the spectrum of clinicopathology, narrowing the spectrum of suspected etiologies. *Chest* 2000;117:251-9.